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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/750,005	12/30/2003	Herbert T. Nagasawa	30451.2USU1	9934

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EXAMINER	
HEARD, THOMAS SWEENEY	
ART UNIT	PAPER NUMBER
1654	

SHORTENED STATUTORY PERIOD OF RESPONSE	MAIL DATE	DELIVERY MODE
3 MONTHS	04/11/2007	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

If NO period for reply is specified above, the maximum statutory period will apply and will expire 6 MONTHS from the mailing date of this communication.

Office Action Summary	Application No.	Applicant(s)	
	10/750,005	NAGASAWA ET AL.	
	Examiner	Art Unit	
	Thomas S. Heard	1654	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *15 February 2007*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-4,7,9,10,20-22,25,26,33-35,38,39,46,47,50 and 51* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-4, 7, 9, 10, 20-22, 25, 26, 33-35, 38, 39, 46, 47, 50, and 51* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

The Applicants Amendments to the claims received on 2/15/2007 is acknowledged. The text of those sections of Title 35 U.S. Code not included in the action can be found in the prior office action. Rejections or objections not addressed in this office action with respect to the previous office action mailed 11/15/2006 are hereby withdrawn.

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

Claims 1-4, 7, 9, 10, 20-22, 25, 26, 33-35, 38, 39, 46, 47, 50, and 51 are rejected under 35 U.S.C. 103(a) as being unpatentable over:

Shirota FN, DeMaster EG, Shoeman DW, Nagasawa HT, "Acetaminophen-induced suppression of hepatic AdoMet synthetase activity is attenuated by prodrugs of L-cysteine," Toxicol Lett. 2002 Jun 7;132(1):1-8;

Jonas AJ et al, "Cystine accumulation and loss in normal, heterozygous, and cystinotic fibroblasts," Proc Natl Acad Sci U S A. 1982 Jul;79(14):4442-5; and

Bender AS et al, "Characterization of cystine uptake in cultured astrocytes," Neurochem Int. 2000 Aug-Sep;37(2-3):269-76.

Applicant's arguments have been carefully considered but have not been found persuasive. Applicants have argued:

The legal standard for a rejection under §103 is as follows. As set forth in MPEP §2143: To establish a prima facie case of obviousness, three basic criteria must be met. First, there must be some suggestion or motivation, either in the references themselves or in the knowledge generally available to one of ordinary skill in the art, to modify the reference or to combine reference teachings. Second, there must be a reasonable expectation of success. Finally, the prior art reference (or references when combined) must teach or suggest all the claim limitations. The teaching or suggestion to make the claimed combination, and the reasonable expectation of success, must both be found in the prior art, not in the Applicants' disclosure (In re Vaeck, 947 F.2d 488, 20 USPQ2d 1438 (Fed. Cir. 1991)).

First, one would have been motivated to administer CYSSG (also called CSSG) Jonas' teaching that intracellular Cystine production can be induced with the administration of CySSG instantly claimed, and with Bender' teaching that Cystine is converted to cysteine, the precursor to GSH and the reduction of oxidative stress. Secondly, there was a reasonable expectation of success because the Applicants elected species has already been administered to cells and Cysteine levels were increased three fold. Thirdly, the prior art references that were combined did teach the present invention as one skilled in the art would recognize the nexus between cysteine production and GSH levels, important for regulating oxidative stress. Finally, the references were not found in the Applicants disclosure but through literature search. None of the references were in the Applicant's IDS.

In response to applicant's argument that the examiner's conclusion of obviousness is based upon improper hindsight reasoning, it must be recognized that any judgment on obviousness is in a sense necessarily a reconstruction based upon hindsight reasoning. But so long as it takes into account only knowledge which was within the level of ordinary skill at the time the claimed invention was made, and does not include knowledge gleaned only from the applicant's disclosure, such a

reconstruction is proper. See In re McLaughlin, 443 F.2d 1392, 170 USPQ 209 (CCPA 1971).

The Applicants argue that the prior art does not teach *the substitution of CySSG for CySSME, much less substitution of CySSG for CYSSME for the production of GSH and the reduction of oxidative stress.* The Examiner agrees that neither reference taught the specific substitution, and if it did may have constituted a 102 rejection. Instead, it was clear from the prior art that it would have been obvious to substitute the naturally occurring CYSSG (CSSG) for the production of GSH through Cysteine production as taught by Bender.

Applicants have further argued:

Shirota et al. teach that (1) three structurally diverse types of L-cysteine prodrugs (NOT sulfhydryl protected glutathione prodrugs), two of them sulfhydryl-protected (e.g., CySSME), can reduce acetaminophen induced hepatic toxicity as measured by plasma alanine aminotransferase (ALT) activity and (2) maintenance of glutathione homeostasis can protect hepatic AdoMet sythetase activity (Abstract). The L-cysteine prodrug CySSME is a mixed disulfide of cysteine and mercaptoethanol and is dependent on enzymatic reduction of its disulfide bond to release eysteine (page 5, 2na column, 3ra paragraph). Shirota et al. also teach that hepatoproteetion by the cysteine generated from various cysteine prodrugs is due to enhanced GSH synthesis and maintenance of hepatic GSH homeostasis (page 5, 2na column, last paragraph). Shirota et al. do not teach use of any sulfhydryl protected glutathione prodrug let alone use of a sulfhydryl protected glutathione prodrug to provide glutathione directly to cells without de novo glutathione synthesis or to reduce oxidative stress in a eel/. L-Cysteine prodrugs and sulfhydryl protected glutathione prodrugs are not equivalents; reduction of sulthydryl protected glutathione prodrugs releases preformed glutathione (as well as L-cysteine in the case of L-CySSG), while L-eysteine prodrugs (such as CySSME) release L-cysteine (specification at page 9, lines 3-6).

The Examiner's position that this use of prodrug is not an informative term because CSSG or CySSG is a natural product found in the cell and not a prodrug. CySSG and

CSSG are cysteine "prodrugs" because they release cysteine. CySSG was used to raise cysteine levels in the cell and CySSG (CSSG) was also used to increase cysteine levels.

Applicants have argued that:

Jonas et al. teach that cysteine-glutathione mixed disulfide (CSSG) treatment of cystinotic fibroblasts, fibroblasts from patients heterozygous for cystinosis and normal (i.e., non-cystinotic) fibroblasts lead to an increase in cystine levels in the cells. Heterozygous mad normal fibroblasts rapidly cleared the accumulated cystine when CSSG treatment was removed while cystinotic fibroblasts retained cystine. (abstract). Jonas et al. teach that CSSG provides "a soluble source of cyst(e)ine" for cells (page 4442, 1st column, 2nd paragraph). Jonas et al. do not teach use of any sulfhydryl protected glutathione prodrug (e.g., CSSG), let alone a sulflaydryl protected glutathione prodrug to release preformed glutathione to cells or to reduce oxidative stress in a cell.

First, it does not matter that cyst(e)ine was rapidly cleared. It was first formed before it was cleared so it increased cysteine levels, important for GSH production. The Applicants are again using the term sulfhydryl protected prodrug in an odd way. It is asserted "*Jonas et al do not teach use of any protected glutathione prodrug (e.g. CSSG)*. The abstract of Jonas reads as follows:

Cystinotic fibroblasts contain approximately 100 times more cystine than do normal control fibroblasts. **When cystinotic fibroblasts were placed in the presence of 30 mM cysteine-glutathione mixed disulfide (CSSG) for 24 hr, their cystine content increased about 3-fold.** Similar treatment of normal fibroblasts and fibroblasts from patients heterozygous for cystinosis resulted in a 6- to 7-fold increase in cystine content. In all three cell types, the intracellular free cystine is located within lysosomes. When placed in cystine-free medium after 24 hr in CSSG-containing medium, the normal and heterozygous fibroblasts rapidly lost their lysosomal cystine (t 1/2 = 20 min), but the cystine content of the cystinotic cells remained stable for over 90 min. In contrast to the findings in intact fibroblasts, cystine loss could not be demonstrated from isolated, cystine-loaded lysosomes from any of the three cell types.

Jonas et al did teach the use of CSSG. Because Jonas did note state a sulfhydryl protected prodrug does not mean that what Jonas administered was not CSSG or CySSG. Jonas et al demonstrated that cysteine was increased in all cells tested, see Table 1. Thus, it follows from Jonas, Bender, and Shirota that GSH would also have been produced.

Applicants have further argued:

Bender et al. studied the mechanism of cystine uptake in astrocytes and found that astrocytes transport cystine through a similar, if" not identical, system used to transport glutamate (Abstract). Bender et al. also teach that cellular uptake of cystine is the rate-limiting step in the biosynthesis of glutathione (Abstract). Cystine, after transport into cells, is reduced to eysteine, a precursor of glutathione. Thus, cystine, via cysteine, is required for maintaining cellular levels of glutathione. Glutathione protects cells against oxidative stress and various toxins. (Abstract) However, Bender et al. do not teach use of any sulflaydryl protected glutathione prodrug, let alone a sulfhydryl protected glutathione prodrug to release preformed glutathione to cells or to reduce oxidative stress in a cell. The Office alleges that Bender's teaching (that cystine is reduced to cysteine which is used to synthesize glutathione) provides a nexus between the administration of CySSG (as taught by Jonas et al.) and the production of GSH for the reduction of oxidative stress with a L-cysteine prodrug (as taught by Shirota et al.) (last paragraph on page 3 of the outstanding Office Action). Thus, the Office concludes that it would have been obvious to substitute CySSG (a sulfhydryl protected glutathione prodrug) for CySSME (a L-cysteine prodrug) (first paragraph on page 4 of the outstanding Office Action).

Given that CySSG and CSSG are mixed disulfides of GSH, once reduced would liberate cysteine and it seems the evidence is in favor that the release of cysteine is an important role in the production and maintenance of GSH. Applicants have pointed out that "*cystine, via cysteine, is required for maintaining cellular levels of glutathione.*" It would seem that this is what CySSG (CSSG) and CySSME were doing, raising the

cysteine levels, which are important in GSH production and is the nexus for the combination of references to make the rejection.

Applicants have further argued:

In order for an obviousness rejection to be proper, the prior art itself must suggest the desirability of making the required modification or combination. As the Court of Appeals for the Federal Circuit has held: The mere fact that the prior art could be so modified would not have made the modification obvious unless the prior arc suggested the desirability of the modification. (**emphasis added**)[1]. Here, no such suggestion is presented by the cited publications. It is clear that "obvious to try" is not the appropriate legal standard. (In re Fine, 837 F.2d 1071, 5 USPQ2d 1596 (Fed. Cir. 1988); In re Geiger, 815 F.2d 686, 2 USPQ2d 1276, 1278 (Fed. Cir. 1987; In re O'Farrell, 853 F.2d 894, 7 USPQ2d 1673 (Fed. Cir. 1988).

The Examiner would also like to contribute the following legal argument to rebut that one needs explicit motivation to combine.

The rationale to modify or combine the prior art does not have to be expressly stated in the prior art; the rationale may be expressly or impliedly contained in the prior art or it may be reasoned from knowledge generally available to one of ordinary skill in the art, established scientific principles, or legal precedent established by prior case law. In re Fine, 837 F.2d 1071, 5 USPQ2d 1596 (Fed. Cir. 1988); In re Jones, 958 F.2d 347, 21 USPQ2d 1941 (Fed. Cir. 1992). See also In re Kotzab, 217 F.3d 1365, 1370, 55 USPQ2d 1313, 1317 (Fed. Cir. 2000) (setting forth test for implicit teachings); In re Eli Lilly & Co., 902 F.2d 943, 14 USPQ2d 1741 (Fed. Cir. 1990) (discussion of reliance on legal precedent); In re Nilssen, 851 F.2d 1401, 1403, 7 USPQ2d 1500, 1502 (Fed. Cir. 1988) (references do not have to explicitly suggest combining teachings); Ex parte Clapp, 227 USPQ 972 (Bd. Pat. App. & Inter. 1985) (examiner must present convincing line of reasoning supporting rejection); and Ex parte Levengood, 28 USPQ2d 1300 (Bd. Pat. App. & Inter. 1993) (reliance on logic and sound scientific reasoning).

It is further argued from Applicants that:

Contrary to the Office's statement on page 4 of the outstanding Office Action, there is no motivation to substitute CySSG for CySSME for reducing oxidative stress in a cell. CySSG and CySSME do not share a common utility mad function (In re Lalu and

Foulletier 747 F.2D 703,223 USPQ 1257 (Fed. Cir. 1984)). CySSG is a sulfhydryl protected glutathione prodrug while CySSME is sulfhydryl protected cysteine prodrugs (specification at page 3, lines 9-12). CySSG is a mixed disulfide of L-cysteine and glutathione. When reduced, CySSG releases a preformed glutathione as well as L-cysteine. The L-cysteine is a precursor for de novo glutathione synthesis. Thus, CySSG produces two glutathiones; the first glutathione is released from CySSG by reduction making it immediately available to the cell; the second glutathione is de novo synthesized by the cell from the L-cysteine that was released by CySSG (specification at page 9, first paragraph). CySSME is a mixed disulfide of L-cysteine and mercaptoethanol. Only a single glutathione can be produced from CySSME, this via de novo synthesis.

Given that CySSG releases Cysteine, it appears that CySSG could be considered a sulfhydryl protected **cysteine** prodrug as well as a sulfhydryl protected **glutathione** prodrug. That fact that the Applicants are arguing a mechanism of how CySSG (CSSG) raised GSH levels, it does not make it patentable because when Jonas et al added CySSG (CSSG) to the cell, it would have been producing GSH along with Cysteine, and the Cysteine it produced would also make GSH. Applicants are arguing a latent property of CySSG (CSSG) to release GSH in addition to Cysteine. CySSG (CSSG) and CySSME both share the common property to release/produce cysteine in the cell, leading to GSH levels which are important for oxidative stress management.

Applicants have argued:

The mere fact that references can be combined does not render the resultant combination obvious unless the prior art also suggests the desirability of the combination. In re Mills, 916 F.2d 680, 16 U.S.P.Q.2d 1430, cited in MPEP §2143.01. There must be a reason or suggestion in the art for modifying the prior art other than the knowledge learned from applicants' disclosure. However, the cited references provide none. The primary reference, Shirota et al., teach that prodrugs of L-cysteine, one of them being CySSME, can protect hepatic AdoMet synthetase activity and reduce acetaminophen induced hepatic toxicity by providing L-cysteine. The secondary references do not teach or suggest what the primary reference fails to teach, namely,

the use of sulfhydryl protected glutathione prodrug (e.g., CSSG) to deliver preformed glutathione to cells or to reduce oxidative stress in a cell. Further, and contrary to the Office's statement, there would have been no motivation to substitute CySSG for CySSME because they are not equivalents. Accordingly, the combination of the primary and secondary references does not and cannot render obvious the claimed methods

Again, the rationale to modify or combine the prior art does not have to be expressly stated in the prior art; the rationale may be expressly or impliedly contained in the prior art or it may be reasoned from knowledge generally available to one of ordinary skill in the art, established scientific principles, or legal precedent established by prior case law. In re Fine, 837 F.2d 1071, 5 USPQ2d 1596 (Fed. Cir. 1988); In re Jones, 958 F.2d 347, 21 USPQ2d 1941 (Fed. Cir. 1992). See also In re Kotzab, 217 F.3d 1365, 1370, 55 USPQ2d 1313, 1317 (Fed. Cir. 2000) (setting forth test for implicit teachings); In re Eli Lilly & Co., 902 F.2d 943, 14 USPQ2d 1741 (Fed. Cir. 1990) (discussion of reliance on legal precedent); In re Nilssen, 851 F.2d 1401, 1403, 7 USPQ2d 1500, 1502 (Fed. Cir. 1988) (references do not have to explicitly suggest combining teachings); Ex parte Clapp, 227 USPQ 972 (Bd. Pat. App. & Inter. 1985) (examiner must present convincing line of reasoning supporting rejection); and Ex parte Levengood, 28 USPQ2d 1300 (Bd. Pat. App. & Inter. 1993) (reliance on logic and sound scientific reasoning). The use of CySSG (CSSG) in place of CySSME does flow logically as both increase Cysteine, and the increase in cysteine leads to increased GSH.

Finally, Applicants argue:

Even if. "Obvious to Try" is the Appropriate Legal Standard, One of Ordinary Skill in the Art would not Expect Successful Results Although obviousness under 35 U.S.C. §103 does not require absolute predictability of success, 35 U.S.C. §103 does require a

reasonable expectation of success to find obviousness. (In re O'Farrell, 853 F.2d 894, 7 USPQ2d 1673 (Fed. Cir. 1988)). Given the state of the art, there would have been no reasonable expectation that one would be able to produce the claimed method by simply substituting a sulfhydryl protected glutathione prodrug for a cysteine prodrug, sulfhydryl-protected or otherwise.

Both CySSME and CYSSG (CSSH) were shown to produce an increase in cysteine levels which are important for producing GSH. Therefore, it worked and was successful. Increasing cysteine levels increase GSH and increasing GSH reduces oxidative stress. CySSG is not a sulfhydryl protected glutathione prodrug, but a natural mixed disulfide between cysteine and GSH. If it releases cysteine, as Applicants argued supra, and it increased cysteine levels in the cell, as shown by Jonas et al, then CySSG must be a sulfhydryl protected cysteine prodrug, and would be an obvious substitute for CySSME.

Therefore, Shirota et al teaches the suppression of hepatotoxicity and oxidative stress induced by acetaminophen by the administration of the prodrug of L-cysteine, specifically that of CySSME. Shirota et al teaches that the "hepatoprotection by cysteine generated from a prodrug, however, is due to enhanced GSH synthesis and maintenance of hepatic GSH homeostasis rather than to direct scavenging of the reactive-ACP metabolite by cysteine, see page 5, second column and last paragraph. Shirota et al does not teach the use of the naturally occurring, mixed-disulfide, L-Cysteine "prodrug" CySSG.

Jonas AJ teaches the administration of CSSG (the same as the Applicant's CySSG) for the induction of Cystine in both normal, heterozygous, and cystinoic

fibroblasts. Bender A.S. et al teaches that "*the amino acid cystine is required for maintaining cellular levels of glutathione, a compound which protects cells against oxidative stress and various toxins (Meister and Anderson, 1983). Once taken up by cells, cystine is reduced to cysteine, the rate-limiting precursor of glutathione synthesis (Bannai and Teteishi, 1986).*"

The difference between what is instantly claimed and the prior art is that Bender AJ provides the nexus between the administration of CySSG and CySSME and the production of GSH for the reduction of oxidative stress with a L-Cysteine producing compound.

It would have been obvious at the time of the instantly claimed invention to substitute CySSG for CySSME for the production of GSH and the reduction of oxidative stress due to the toxic dose of acetaminophen. One would have been motivated to do so given Jonas' teaching that intracellular Cystine production can be induced with the administration of CySSG instantly claimed, and with Bender' teaching that Cystine is converted to cysteine, the precursor to GSH. From the teachings of the references, it is apparent that one of ordinary skill in the art would have had a reasonable expectation of success in producing the claimed invention. Therefore, the invention as a whole was *prima facie* obvious to one of ordinary skill in the art at the time the invention was made, as evidenced by the references.

Conclusion

No claims are allowed.

THIS ACTION IS MADE FINAL. Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the mailing date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Thomas S. Heard whose telephone number is (571) 272-2064. The examiner can normally be reached on 9:00 a.m. to 6:30 p.m..

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Cecilia Tsang can be reached on (571) 272-0562. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should

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TSH

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